



## King's Research Portal

DOI:

[10.1097/PSY.0000000000000327](https://doi.org/10.1097/PSY.0000000000000327)

*Document Version*

Peer reviewed version

[Link to publication record in King's Research Portal](#)

*Citation for published version (APA):*

Pick, S., Mellers, J. DC., & Goldstein, L. H. (2016). Explicit facial emotion processing in patients with dissociative seizures. *Psychosomatic Medicine*, 78(7), 874-885. <https://doi.org/10.1097/PSY.0000000000000327>

### **Citing this paper**

Please note that where the full-text provided on King's Research Portal is the Author Accepted Manuscript or Post-Print version this may differ from the final Published version. If citing, it is advised that you check and use the publisher's definitive version for pagination, volume/issue, and date of publication details. And where the final published version is provided on the Research Portal, if citing you are again advised to check the publisher's website for any subsequent corrections.

### **General rights**

Copyright and moral rights for the publications made accessible in the Research Portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognize and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the Research Portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the Research Portal

### **Take down policy**

If you believe that this document breaches copyright please contact [librarypure@kcl.ac.uk](mailto:librarypure@kcl.ac.uk) providing details, and we will remove access to the work immediately and investigate your claim.

**REVISION**

**Explicit facial emotion processing in patients with dissociative seizures**

**Susannah Pick (PhD)<sup>1</sup>, John D.C. Mellers (MRCPsych)<sup>2</sup>, & Laura H. Goldstein  
(PhD)<sup>1</sup>**

<sup>1</sup>. King's College London, Department of Psychology, Institute of Psychiatry, Psychology, & Neuroscience, London, UK

<sup>2</sup>. Neuropsychiatry Department, Maudsley Hospital, South London and Maudsley NHS Foundation Trust, London, UK

**Correspondence to:** Prof Laura H Goldstein, Dept of Psychology, PO77, Institute of Psychiatry, Psychology & Neuroscience, King's College London, De Crespigny Park, London, SE5 8AF. Email: [laura.goldstein@kcl.ac.uk](mailto:laura.goldstein@kcl.ac.uk). Telephone ++44 (0)207 848 0218. Fax ++44 (0)207 848 5006

Running head: Emotion recognition in dissociative seizures

Word count: 7887

Figures: 0

Tables: 4

### **Conflicts of Interest and Sources of Funding**

The research was funded by a postgraduate studentship awarded to SP from the Department of Psychology, Institute of Psychiatry, Psychology and Neuroscience (King's College London), and a grant from the Central Research Fund (University of London). This paper also represents independent research part-funded (LHG) by the National Institute for Health Research (NIHR) Dementia Biomedical Research Unit at South London and Maudsley NHS Foundation Trust and King's College London. LHG and JDCM are in receipt of further funding from the NIHR. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health.

No conflicts of interest are declared.

## ABSTRACT

**Objective:** There have, as yet, been few experimental studies of explicit facial affect recognition in patients with dissociative seizures (DS). The aim of the study was to examine explicit recognition and physiological responses to facial expressions in this group, relative to healthy controls.

**Methods:** Forty patients with DS and 43 controls completed a computerised test of facial affect recognition, including five basic expressions (happiness, anger, disgust, fear, neutral). Recognition accuracy, emotional intensity judgements, skin conductance levels (SCLs) and responses (SCRs) were dependent measures. Analyses controlled for a range of potentially confounding variables, including anxiety, depression and medication effects.

**Results:** The DS group were less accurate at identifying facial expressions than controls ( $p = .005$ ,  $\eta^2_p = .10$ ). No group difference emerged for intensity judgements ( $p = .72$ ,  $\eta^2_p = .002$ ). Mean SCLs were higher in the DS group relative to controls ( $p = .046$ ,  $\eta^2_p = .053$ ). However, a subgroup of DS patients showed attenuated SCRs to the facial stimuli, compared to controls ( $p = .015$ ,  $\eta^2_p = .18$ ). These differences could not be accounted for by possible confounding variables. Recognition accuracy for neutral faces correlated negatively with trauma scores ( $r = -.486$ ,  $p = .002$ ) and abandonment concerns ( $r = -.493$ ,  $p = .002$ ) in the DS group.

**Conclusions:** Patients with DS showed reduced recognition accuracy for facial affect, despite accurately perceiving its intensity. Elevated autonomic arousal may characterise patients with DS in general, alongside reduced phasic autonomic responses to facial expressions in some patients with the disorder.

**Key words:** dissociative seizures, psychogenic non-epileptic seizures, facial expression, emotion recognition, skin conductance, autonomic response

**Abbreviations:** DS = dissociative seizures; YoE = years of education; AEDs = antiepileptic drugs; SCLs = skin conductance levels; SCRs = skin conductance responses; WASI = Wechsler Adult Intelligence Scale; FSIQ = Full-scale IQ; BFRT = Benton Facial Recognition Test; WMS-III = Wechsler Memory Scale (3<sup>rd</sup> ed); HADS = Hospital Anxiety and Depression Scale; TEC = Traumatic Experiences Checklist; IASC = Inventory of Altered Self-Capacities;  $\mu$ S = microSiemens; IQR = interquartile range; SD = standard deviation; SE = standard error; mdn = median

## **INTRODUCTION**

Dissociative seizures (DS) are transient alterations in awareness, behaviour and voluntary control, outwardly resembling epileptic seizures but not accompanied by electrographic epileptiform activity, nor better explained by another specific medical/psychiatric diagnosis. Instead, DS are thought to be a result of psychological mechanisms involving abnormal emotional processes (1-4). DS represents a disorder that spans the interface between psychology, neurology and psychiatry, including somatic, cognitive, affective and functional neurological manifestations, without known organic explanation. As such, the disorder is of interest to professionals working within services for people with functional neurological disorders, or medically unexplained symptoms more generally.

DS are also commonly referred to as conversion, functional, or (psychogenic) non-epileptic seizures. The term DS is adopted here in accordance with the current international classification system (5), in which DS are classified alongside other conversion disorders within the dissociative disorders group. The disorder is differentially classified as one of the somatic symptom disorders group in DSM-5, in addition to other disorders indicative of physical illness without any discernible organic causation (6). This discrepancy has been debated by experts in the field, who have advocated the reclassification of DS (and other conversion disorders) with the dissociative disorders in DSM revisions (7, 8). The reconceptualisation of DS as dissociative is primarily based on the presence of shared risk factors with dissociative disorders (9), elevated rates of dissociative symptoms/disorders in patients with DS (2, 10, 11), and importantly, the occurrence of marked losses in integration of usually integrated psychological and somatic processes during the events. Moreover, a

dissociative model of DS can provide patients and carers with a clearer understanding of the possible psychological processes that might underlie their seizures.

Patients with DS report high rates of childhood abuse (12), stressful circumstances (13), and relationship dysfunction (14). A range of emotional difficulties such as anxiety, depression (15, 16), and emotion dysregulation (17) are also common. The presence of maladaptive coping styles (18) and elevated dissociative (19) and somatoform symptoms (20) also suggest aberrant responses to emotional distress in this group. Despite the abundance of observational research and theoretical literature suggesting emotional dysfunction as a key characteristic of patients with DS, it is only relatively recently that emotional processes have been examined in this group using laboratory-based experimental methods.

Bakvis and colleagues (21) provided empirical evidence for alterations in preconscious attentional allocation towards negative emotional facial expressions (i.e. anger) in patients with DS, relative to healthy controls. Subsequent studies have also suggested behavioural avoidance of negative facial expressions (22), increased cognitive interference by emotional faces (23, 24), and differences in the intensity of subjective emotional reactions to general emotional images (25).

Impairments in explicit facial affect recognition have been observed in a range of other disorders sharing clinical characteristics with DS, including depersonalisation disorder (26), borderline personality disorder (27), post-traumatic stress disorder (28) and mixed somatoform diagnoses (29). However, there is currently only one published study of explicit facial expression recognition in patients with DS. Schönenberg et al.

(30) reported that patients with DS had intact facial expression detection abilities relative to healthy controls, when presented with dynamic facial displays gradually changing from neutral to one of the six 'basic' facial expressions. However, this study was likely to be underpowered statistically and a proportion of the patient sample had potentially confounding diagnoses of anxiety or depression. Therefore, further research on this topic is needed, utilising larger samples, additional tests of facial affect recognition, and controlling for the possible influence of important confounding variables.

The current study was designed to assess explicit recognition, perception of emotion intensity and autonomic responses to emotional facial expressions in this group, relative to healthy individuals. On the basis of previous literature, it was predicted that the DS group would show reduced recognition accuracy, alongside heightened perceptions of emotional intensity for facial expressions. On the basis of recent models of DS, suggesting an important role for heightened affective arousal in the seizures (1, 2), and observations of increased sympathetic arousal and cortisol levels more generally (21, 25, 31, 32), it could also be predicted that the DS group would exhibit elevated skin conductance levels (SCLs), and increased skin conductance responses (SCRs) to the stimuli, relative to controls.

## **METHODS**

The study was approved by the Joint South London and Maudsley and Institute of Psychiatry NHS Research Ethics Committee (reference 08/H0807/82). All participants provided written informed consent prior to participation.



## **Participants**

Recruitment and data collection took place between January 2009 and December 2012. Patients were recruited from tertiary care neuropsychiatry clinics (South London and Maudsley NHS Foundation Trust). Recruitment of control participants was carried out via local advertisement. Eligibility criteria for both groups included being 18-65 years old, without documented intellectual disability, and being fluent in English.

For the patient group, diagnosis of DS was confirmed by video-EEG or consensus clinical opinion. Video-EEG diagnosis required the video-recording of at least one typical seizure in the absence of clear epileptogenic EEG discharges during inpatient monitoring. Consensus clinical opinion (agreement by at least two neurologists, or a neurologist and neuropsychiatrist) was determined on the basis that a confident diagnosis of DS (and no suspicion of comorbid epilepsy) could be made on clinical grounds alone and that video-telemetry was unnecessary. This process reflects routine clinical practice at our centre. Most patients in the sample had undergone one or more diagnostic tests, such as routine-EEG, structural neuroimaging, and/or video-EEG (see Results).

Comorbid diagnoses of major neurological conditions (e.g. epilepsy), depression, anxiety, substance dependence, or psychosis were excluded from both samples. Patients with DS who had a history of depression/anxiety disorders and whose symptoms were currently in remission were included in the study. Patients with DS had not undergone psychological treatment for DS. Any current psychiatric or medical diagnoses were exclusion criteria for control participants.

## **Facial emotion processing task**

### **Materials**

Facial stimuli were taken from a standardised set (33), selected on the basis of normative ratings (i.e. those with the highest recognition rates were included). Full details of the stimuli can be found in Supplemental Digital Content 1. The images were digitally cropped to maximise allocation of attention to the emotional expressions. The experimental task was programmed with E-Prime experimental software (Psychology Software Tools, Inc).

### **Design**

The experiment had a mixed factorial design, with one between-groups factor (diagnostic status: DS, control) and one within-groups factor (facial expression: anger, happiness, fear, disgust, neutral). The dependent measures were recognition accuracy (selection of an emotional descriptor from a list of five options, as above), ratings of emotion intensity (0-7 Likert scale; no emotion-very strong emotion), and SCRs (frequency, amplitude). Six examples of each facial expression yielded a total of 30 trials. Stimuli were presented in a novel pseudo-random order for each participant.

### **Procedure**

Prior to commencing the experiment, written standardised instructions were presented on screen, and participants had the opportunity to complete three practice trials. Experimental trials commenced with a 15-second central fixation cross, followed by presentation of an individual face showing one of the target emotions for six seconds.

On stimulus offset, participants were required to select the emotional descriptor that best described the facial emotion, and then rate its intensity.

#### Skin conductance (SC) measurement

Procedures for SC acquisition, reduction and analysis followed well-accepted published guidelines (34, 35). Details are provided in Supplemental Digital Content 2. SC was recorded during a five-minute baseline habituation period and continuously throughout the experimental task. Mean SCLs were calculated separately for these time points. Positive SCRs ( $> .01$  microSiemens,  $\mu\text{S}$ ) occurring during facial stimulus presentation were interpreted as reflecting arousal responses to the stimuli. Maximum amplitude values were calculated relative to pre-stimulus baseline measurements for each stimulus and averaged by condition.

#### **Emotion comprehension check**

Participants were asked to describe each of the five target emotional states verbally, or to provide an example of a situation that might trigger the emotional state, in order to ensure adequate comprehension of the emotion descriptor labels.

#### **Cognitive tests**

Current intellectual functioning was assessed using the two subscale (Vocabulary, Matrix Reasoning) form of the Wechsler Abbreviated Scale of Intelligence (36) (WASI), in order to confirm the absence of intellectual deficits in either group. To evaluate potential cognitive confounds on the experimental task, general perceptual processing of facial stimuli was measured using the short-form version of the Benton Facial Recognition Test (37) (BFRT). The test involves matching concurrently presented faces on the basis of identity and was included to ensure that any group

differences could not be accounted for by differences in basic perceptual processing of facial stimuli. In addition, short-term memory for facial stimuli (recognition) was measured using the Faces 1 subtest of the Wechsler Memory Scale – Third Edition (38) (WMS-III), due to the task demand of providing responses at offset of each facial stimulus.

### **Self-report questionnaires**

Self-report questionnaires were administered to measure psychosocial variables of possible relevance to experimental task performance. These were the Hospital Anxiety and Depression Scale (39), Traumatic Experiences Checklist (40), and the Inventory of Altered Self-Capacities (41).

#### **Traumatic Experiences Checklist (TEC) (40)**

The TEC assesses the presence of 29 types of potentially traumatic experiences. From this measure, it was possible to record the total number of traumatic experiences participants reported (0-29) and also to examine the perceived impact of those experiences (1-5; none-extreme).

#### **Hospital Anxiety and Depression Scale (HADS) (39)**

This reliable and valid scale (39, 42) was developed for use in general medical settings, measuring current (non-somatic) symptoms of anxiety and depression over the previous week. The scale consists of 14 items, with seven items examining anxiety and seven measuring depression. Both subscales have a maximum score of 21, with scores of 8-10 identifying borderline/doubtful cases, and scores of 11-21 indicative of definite 'caseness'.

### Inventory of Altered Self-Capacities (IASC) (41)

This scale which has good reliability and validity (41, 43) assesses difficulties in the maintenance/regulation of identity, emotions, relationships and behaviour. The IASC provides scores on seven subscales which can be categorised into three groups, as follows:

#### 1. Relatedness

- i. Interpersonal Conflict: Reports of problems in relationships.
- ii. Idealisation-Disillusionment: Ambivalent feelings towards significant others.
- iii. Abandonment Concerns: Sensitivity to abandonment by others (real, perceived).

#### 2. Identity

- i. Identity Impairment: Difficulties in maintaining a stable experience of personal identity and self, across time and situations.
- ii. Susceptibility to Influence: Tendencies towards being guided or unduly influenced by others.

#### 3. Affect control

- i. Affect Dysregulation: Deficits in control and regulation of affect.
- ii. Tension Reduction Activities: Externalisation of emotional distress.

Each IASC subscale comprises nine items, which are scored from 1-5. Respondents indicate the frequency of each symptom in the previous six months. Therefore, the

total score for the scale is between 9 and 45. These scores are then converted to T-scores.

### **Statistical analyses**

Statistical analyses were carried out using SPSS (IBM, version 22). Outliers were identified on the basis of z-scores  $>3.29$  (44). Analyses were run with and without outlying scores; if different results were obtained, the results without outliers are presented.

Mean recognition accuracy scores (0-6), intensity ratings (0-7), SCR amplitudes ( $\mu\text{S}$ ), and the proportion of positive SCRs (%) were entered as the dependent variables in a series of factorial ANCOVAs. Group (DS, control) was entered as the between-groups factor and facial expression (happiness, anger, fear, disgust, neutral) as the within-subjects factor. For SCLs, group (DS, control) was the between-subjects factor and time (baseline, task) was the within-subjects factor. Years of education (YoE) and/or anxiety and depression scores (HADS) were included as covariates in these analyses, because significant between-group differences were observed on these variables. Where relevant, analyses were run twice more with AEDs or antidepressants added as additional between-group factors, in order to assess possible medication effects. The decision to explore the effects of these possible confounds was made a priori, to improve the interpretability of participants' performance on the experimental task.

The hierarchical analysis strategy was based on the following power calculation: for a two group ANCOVA, at  $p = .05$ , a total sample of 74 was estimated to have 80%

power to detect a medium effect size of .5 (45). Partial eta squared ( $\eta^2_p$ ) values are presented to illustrate estimated effect sizes for main effects and interactions. According to Cohen (46), rules of thumb for interpretation of eta squared effect sizes are as follows: small (.01), medium (.06), and large (.14).

Parametric tests were conducted with normally distributed data and non-parametric tests or robust parametric tests (i.e. ANCOVA) with non-normal data. Pearson's or Spearman's correlations were carried out to examine relationships between the experimental dependent measures, and between the experimental measures and scores on the self-report questionnaires. Chi-square tests were used to analyse categorical variables. When multiple tests were conducted with related variables (e.g. subscales of self-report measures; post-hoc tests; exploratory correlational analyses), a more stringent alpha level of  $p < .01$  was adopted to reduce the likelihood of Type 1 errors.

## **RESULTS**

### **Participant characteristics**

The two groups were well matched on most demographic variables with the exception of YoE (Table 1), which was lower in the DS sample. The most common medications in the DS group were anti-epileptic drugs (AEDs;  $n = 17$ ; 43%) and antidepressants ( $n = 16$ ; 40%). Patients were taking AEDs due to having only recently been diagnosed with DS and were being tested during dosage reduction, or were taking AEDs for mood stabilisation in a minority of cases. Antidepressants were prescribed for comorbid anxiety or affective symptoms. Most patients (68%) were diagnosed on the basis of video-EEG, with the remainder (32%) diagnosed on the basis of clinical

consensus. However, other diagnostic tests were very common in all patients (Table 1).

Table 1 near here

### **Emotion comprehension check**

All participants were able to provide definitions of the five emotion labels, or gave relevant examples of situations that might trigger or be associated with each emotion.

### **Cognitive tests**

Scores for the cognitive tests are shown in Table 2. The groups were well-matched on the WASI and WMS-III Faces tests, but there was a borderline significant between-group difference on the BFRT, with the DS group performing worse than the control group. A small number of participants in each group scored within the borderline/impaired range on this measure (control  $n = 1$ ; DS  $n = 4$ ). With these participants excluded from the analysis, no group differences were observed in BFRT scores, or any other cognitive measure administered.

Table 2 near here

### **Self-report measures**

Table 3 displays scores obtained on the HADS, TEC and IASC. Patients with DS received significantly higher scores than controls on both subscales of the HADS, total trauma and trauma impact scores on the TEC, and on several subscales of the IASC



(Abandonment Concerns, Identity Impairment, Affect Dysregulation, and Tension Reduction Activities).

Table 3 near here

### Recognition accuracy

Descriptive statistics for emotion recognition accuracy are shown in Table 4. There was a significant main effect of expression ( $F(3.38, 250.4) = 4.19, p = .005, \eta^2_p = .054$ ) and group ( $F(1, 74) = 8.56, p = .005, \eta^2_p = .10$ ). Estimated marginal means indicated inferior performance in the DS group. These main effects remained significant when participants scoring in the borderline or impaired range on the BFRT were removed from the analysis (expression  $p = .014$ , group  $p = .007$ ).

Table 4 near here

Regarding the effect of expression, neutral faces were recognised least accurately (marginal mean = 4.82, SE = .13) and happy faces were recognised most accurately (marginal mean = 5.87, SE = .037). There was no group x expression interaction ( $F(3.38, 250.4) = .69, p = .57, \eta^2_p = .009$ ), indicating the same overall pattern of responses in both groups. YoE ( $F(1, 74) = 2.17, p = .15, \eta^2_p = .029$ ), HADS Anxiety ( $F(1, 74) = 1.13, p = .29, \eta^2_p = .015$ ) and HADS Depression scores ( $F(1, 74) = .25, p = .62, \eta^2_p = .003$ ) were not significant covariates in the analysis. Post-hoc tests indicated that group differences were not significant for any single emotional expression (anger  $p = .070$ ; disgust  $p = .15$ ; fear  $p = .34$ ; happiness  $p = .89$ ; neutral  $p = .13$ ).

AED and antidepressant use were entered next as additional between-group factors in two further runs of the ANCOVA. The group effect remained significant in each case (AEDs:  $F(1, 73) = 6.1$ ,  $p = .016$ ,  $\eta^2_p = .077$ ; antidepressants:  $F(1, 73) = 6.94$ ,  $p = .010$ ,  $\eta^2_p = .087$ ), with the DS group showing worse performance in both analyses. The effect of neither drug class was significant (AEDs:  $F(1, 73) = .87$ ,  $p = .36$ ,  $\eta^2_p = .012$ ; antidepressants:  $F(1, 73) = .057$ ,  $p = .81$ ,  $\eta^2_p = .001$ ).

### **Emotional intensity ratings**

Descriptive statistics for intensity ratings are shown in Table 4. For perceived intensity, there was a significant effect of expression ( $F(1.74, 134.2) = 7.22$ ,  $p = .002$ ,  $\eta^2_p = .086$ ), but no main effect of group ( $F(1, 77) = .13$ ,  $p = .72$ ,  $\eta^2_p = .002$ ), and no group x expression interaction ( $F(1.74, 134.2) = 2.34$ ,  $p = .11$ ,  $\eta^2_p = .029$ ). Neither HADS Anxiety ( $F(1, 77) = .030$ ,  $p = .86$ ,  $\eta^2_p = .000$ ) nor Depression scores ( $F(1, 77) = .091$ ,  $p = .76$ ,  $\eta^2_p = .001$ ) were significant covariates; however, YoE was significant ( $F(1, 77) = .46$ ,  $p = .034$ ,  $\eta^2_p = .057$ ). With reference to the significant expression effect, the perceived intensity in neutral expressions (marginal mean = 1.93, SE = .24) was lowest and the perceived intensity of fear (marginal mean = 5.74, SE = .11) was rated highest. Greater YoE was associated with perceptions of lower intensity in all conditions.

### **Skin conductance measures**

SC data for four control participants were excluded due to technical failures or participants' behaviour (e.g. excessive movement artefact). Table 4 displays descriptive statistics for all SC measures.

### Skin conductance levels (SCLs)

There was a significant effect of time (baseline, task) on mean SCL levels ( $F(1, 75) = 9.53, p = .003, \eta^2_p = .11$ ), with higher values observed in the task relative to the baseline measurements. However, there was no main effect of group ( $F(1, 75) = 3.13, p = .081, \eta^2_p = .040$ ) and no group x time interaction ( $F(1, 75) = .16, p = .69, \eta^2_p = .002$ ). HADS Depression scores were a significant covariate ( $F(1, 75) = 4.92, p = .030, \eta^2_p = .062$ ); however, HADS Anxiety scores were not ( $F(1, 75) = .040, p = .842, \eta^2_p = .001$ ). Higher Depression scores were associated with lower SCLs.

When AEDs were entered into the model as an additional factor, the pattern of findings remained unaltered and AEDs did not exert a significant effect ( $F(1, 74) = .56, p = .46, \eta^2_p = .008$ ). However, with antidepressants entered into the model, the group effect was significant ( $F(1, 74) = 4.12, p = .046, \eta^2_p = .053$ ), although the effect of antidepressants was not ( $F(1, 74) = 1.07, p = .304, \eta^2_p = .014$ ). The DS group had higher mean SCLs (marginal mean = 7.61, SE = .93) than controls (marginal mean = 5.38, SE = .92). HADS Depression scores showed a trend towards significance as a covariate in the latter analysis ( $F(1, 74) = 3.61, p = .061, \eta^2_p = .046$ ). Post-hoc tests, controlling for depression and antidepressant medication, showed that the group difference was significant at baseline ( $p = .024, \eta^2_p = .026$ ) but not during the task ( $p = .071, \eta^2_p = .043$ ).

### Proportion of positive SCRs

There were no significant main effects of group ( $F(1, 74) = .062, p = .80, \eta^2_p = .001$ ) or facial expression ( $F(4, 296) = 1.52, p = .19, \eta^2_p = .020$ ) on the percentage of trials

on which positive SCRs were observed. The interaction between facial expression and group was also non-significant ( $F(4, 296) = .41, p = .81, \eta^2_p = .005$ ). YoE ( $F(1, 74) = 1.03, p = .31, \eta^2_p = .014$ ), HADS Depression ( $F(1, 74) = 1.52, p = .22, \eta^2_p = .020$ ), and HADS Anxiety scores ( $F(1, 74) = .005, p = .94, \eta^2_p = .000$ ) were not significant covariates. Neither AEDs ( $F(1, 73) = .91, p = .34, \eta^2_p = .12$ ) nor antidepressants ( $F(1, 73) = .22, p = .64, \eta^2_p = .003$ ) were significant when added to the model in additional runs of the analysis, neither did their addition alter the above pattern of findings. Amplitude

SCR amplitudes are calculated on the basis of positive responses only; therefore, the analysis of amplitude data was carried out with a reduced sample (DS  $n = 16$ ; control  $n = 16$ ) of 'autonomic responders', defined as those participants showing at least one positive SCR in every condition. There was no group difference in the proportion of participants classified as 'autonomic responders' ( $\chi^2(1, 79) = .010, p = .92$ ). There were also no significant group differences in age ( $t(30) = -.24, p = .81$ ) or gender ( $p = 1$ , Fisher's Exact Test), in the 'autonomic responders' subsample.

For this subgroup of participants, there was no main effect of facial expression ( $F(3.2, 86.4) = .42, p = .75, \eta^2_p = .015$ ) and no group x expression interaction ( $F(3.2, 86.4) = 1.65, p = .18, \eta^2_p = .058$ ) for SCR amplitudes. However, there was a borderline significant main effect of group ( $F(1, 27) = 4.04, p = .055, \eta^2_p = .13$ ), with the DS patients showing a trend towards reduced SCR amplitudes relative to controls. YoE ( $F(1, 27) = .031, p = .86, \eta^2_p = .001$ ), HADS Anxiety ( $F(1, 27) = .33, p = .57, \eta^2_p = .012$ ) and Depression ( $F(1, 27) = .022, p = .88, \eta^2_p = .001$ ) were not significant covariates.

This analysis was possibly underpowered as a result of the reduced sample size, so it was run again with non-significant covariates removed. In this second analysis, the group main effect was significant ( $F(1, 30) = 6.71, p = .015, \eta^2_p = .18$ ), again showing reduced SCRs in the DS group. All participants in this subgroup had scores in the normal range on the BFRT; therefore, this effect could not be explained by differences in visual processing of the faces. However, there was still no significant main effect of facial expression ( $F(2.54, 76.2) = .48, p = .67, \eta^2_p = .016$ ), or group  $\times$  facial expression interaction ( $F(2.54, 76.2) = 1.96, p = .14, \eta^2_p = .061$ ). Post-hoc tests failed to reveal between-group differences in SCR for any single emotion at the required stringent alpha level of  $p < .010$  (anger  $p = .096$ ; disgust  $p = .48$ ; fear  $p = .12$ ; happiness  $p = .024$ ; neutral  $p = .40$ ), although the difference for happy expressions approached significance.

The group effect remained significant when AED and antidepressant use were added to the model in two additional runs of the analysis (AEDs:  $F(1, 29) = 4.7, p = .038, \eta^2_p = .14$ ; antidepressants:  $F(1, 29) = 5.15, p = .031, \eta^2_p = .15$ ). Neither medication class yielded a significant main effect (AEDs:  $F(1, 29) = .28, p = .60, \eta^2_p = .010$ ; antidepressants:  $F(1, 29) = .20, p = .66, \eta^2_p = .007$ ).

### Exploratory correlations

Recognition accuracy scores for neutral faces were negatively correlated with IASC Abandonment Concerns ( $r = -.493, p = .002$ ) and TEC total scores ( $r = -.486, p = .002$ ). These associations remained significant after controlling for YoE with partial correlations ( $p < .01$ ), and were not significant in the control group.

The remaining correlations between the experimental dependent variables and the other subscales of the TEC and IASC either did not meet the alpha level adopted in this study ( $p < .01$ ), and/or or did not survive correction for YoE in partial correlations.

## **DISCUSSION**

This study sought to investigate explicit recognition and autonomic responding to emotional facial expressions, in individuals with DS relative to healthy controls. The findings suggest that individuals diagnosed with DS are less accurate than healthy individuals in categorising the emotional meaning of others' facial expressions. This effect could not be accounted for by the presence of anxiety, depression, cognitive deficits or medication effects, in this sample. The absence of significant interaction effects for recognition accuracy indicated that the overall pattern of responses was similar in both groups. Indeed, the post-hoc tests suggested that the deficit was not specific to any single emotion, but was a result of generally reduced performance across conditions in the DS group. This finding contrasts with Schönenberg et al's study (30), in which patients with DS performed comparably to controls on a test of dynamic facial expression detection. This discrepancy is likely due to methodological differences such as experimental paradigm, sample sizes, and control for possible confounding variables.

Nevertheless, if replicated, the present finding of impaired recognition accuracy may be both theoretically and clinically significant. Difficulties in decoding facial expressions are likely to negatively affect relationships and daily interactions. This impairment may contribute to the heightened emotional distress and relationship

disturbances reported by patients with DS, thereby acting as either a predisposing characteristic and/or a maintaining/triggering factor in the disorder. In addition, misinterpretation of social signals might be associated with therapeutic difficulties such as poor engagement or misunderstandings within the patient-clinician relationship. Clinicians working with this group might find it beneficial to be mindful of the potential for misunderstanding of emotional expressions, and to minimise the use of ambiguous non-verbal behaviours. Moreover, novel psychological interventions aimed specifically at improving emotional interpretation and understanding could be an important direction for the development of treatments for this disorder.

There was an overall expression effect on recognition accuracy, finding that happiness was most easily recognised and neutral least, across all participants. This is in accordance with other studies, which have shown that happiness is generally well-recognised (47) and agreed upon in cross-cultural studies (48). In contrast, as neutral expressions are inherently more ambiguous than expressions of specific emotions, it follows that they might be more difficult to categorise.

The significant expression effect for intensity ratings showed that neutral faces were rated as least intense, again, as might be expected given the lack of emotional arousal being displayed in such expressions. Fear, on the other hand, was rated as most intense, possibly reflecting the known association between fearful face perception and autonomic and limbic responses (49, 50). However, the lack of significant group effects for emotional intensity ratings indicated that patients were accurate in gauging the degree of emotional arousal shown in the faces. The significant covariance of YoE with intensity ratings indicated that across the sample, the more highly educated participants perceived less intensity in the faces. One

explanation could be increased cognitive regulation of responses to affective stimuli in those with more extensive educational history.

The finding that SCL increased between baseline and the experimental task across groups is as would be expected, and might be related to energy utilisation, allocation of attentional resources and/or the effects of affect/stress during psychological task performance (34). Furthermore, the SCL findings provided additional evidence for heightened tonic levels of autonomic arousal in patients with DS, relative to controls. This supports previous findings utilising heart rate variability and cortisol measures of sympathetic activity at rest (25, 31, 51). The current study not only provided further evidence of increased general arousal levels at baseline in patients with DS, but importantly, controlled for the possible influence of depression and antidepressant medication, which are known to be associated with alterations on related measures (52-54). It is possible that this elevated physiological autonomic arousal could in some way contribute to the occurrence of seizures in patients with DS. Indeed, some studies have shown that sympathetic activity is lowered peri-ictally, relative to those with epilepsy (55-57), suggesting that DS may be associated with a temporary reduction of ongoing heightened autonomic arousal.

In contrast to the tonic SC measures, the current study did not support the prediction that elevated autonomic responding to emotional facial stimuli would be observed in the DS group. The proportion of trials on which positive SCRs occurred did not differ between groups, neither did the proportion of each group who responded consistently across all conditions (i.e. 'autonomic responders'). Therefore, in this sample, the DS patients were just as likely to respond to the facial stimuli with autonomic responses



as healthy individuals. The lack of group differences in SCLs during the task also suggests that the two groups were comparable in their overall arousal levels, during the experimental task.

However, for those participants classified as 'autonomic responders', those with DS showed reduced SCRs to the facial stimuli, relative to controls. This was a relatively large effect, and so once again, this finding is likely to reflect an important and possibly clinically relevant feature of this subgroup. The lack of significant group effects for any individual expression condition indicates that the reduced responding was a more general characteristic of autonomic responses to facial emotion in this subsample. However, the trend towards significance for happy expressions is suggestive of a particularly prominent reduction in autonomic responsivity towards positive facial emotion. Hypothetically, reduced physiological reactivity towards positive facial expressions in some patients with DS could reflect affective blunting to such positive social signals, perhaps linked to subjective misinterpretation of these expressions as negative or neutral. Further exploration of responses to positive emotional expressions in future studies seems warranted.

The pattern of reduced SCRs in this subgroup of patients is similar to that observed previously in depersonalisation disorder (58). Patients with DS and depersonalisation disorder both present with frequent, intense and distressing dissociative symptoms (2, 19, 59). Therefore, reduced autonomic responding to emotional faces could be part of a dissociative response to potentially threatening or distressing social cues, or a more general disengagement from external stimuli, in some patients with DS.

Alternatively, it is possible that the observed differences might reflect intentional cognitive avoidance of the stimuli. In social anxiety, for example, it has been found that patients avert their gaze from the eye region of facial stimuli (60). Patients with DS might experience directly-viewed facial expressions as anxiety-provoking and therefore avoid allocating attention to key regions such as the eyes, when the faces are processed explicitly. Such a tendency could compromise the ability to accurately label the faces, and might lead to attenuation of SCRs in some patients with DS. One means of examining this hypothesis would be to replicate the current study including a measure of eye gaze during the experimental task.

Another possible explanation for the recognition accuracy and/or SC findings might be that the DS patients included in the study exerted less effort during the experimental task than controls. There has been some literature suggesting that poor performance on cognitive tests in this group might be explained by reduced effort (61). However, this explanation does not adequately explain the specificity of the deficits to recognition accuracy, as it might be expected that reduced effort would also lead to poor performance for intensity judgements and/or on the standardised cognitive tests administered here. Future studies might seek to incorporate a measure of effort within the test battery to exclude this possible explanation.

In the present study, recognition accuracy for neutral faces was negatively associated with trauma scores in the DS patients. Traumatic experiences involving interpersonal relationships (e.g. abuse), which are known to be common in DS, might be associated with a lack of consistency between others' facial expressions and their behaviour, or between others' facial expressions and the emotional consequences for the observer.

If such experiences occurred during development, they might result in facial expressions being perceived as unpredictable and inconsistent, or as signalling threat when none is present. Interestingly, facial expression recognition deficits in individuals with borderline personality disorder have also been reported to be associated with childhood trauma (62). Moreover, Bakvis et al (21) noted a positive relationship between the experience of sexual abuse and preconscious attentional bias for angry facial expressions in DS. The negative relationship between neutral emotion recognition and abandonment concerns observed in the DS group also suggests that misinterpreting neutral faces is linked with maladaptive relationship schemata. Future research might aim to further elucidate the possible contribution of traumatic life events and associated schemata on social-emotional processing in this group.

The results on the self-report measures presented here support and extend previous findings. For example, the current study replicates the finding that patients with DS report more symptoms of anxiety and depression than healthy individuals, albeit in the normal-borderline ranges in this sample. Higher rates of traumatic experiences (TEC total scores) were also observed, and the study has also shown that this group perceived such adverse life events to have had greater impact on them than controls. This suggests that it is not necessarily just the number of life events experienced, but their psychological consequences that may be an important factor for individuals diagnosed with DS. This suggests the importance of exploring the psychological consequences of traumatic life events in psychological interventions for patients with DS who report such experiences.

Moreover, the results on the IASC provide further evidence that individuals with DS experience a range of difficulties in personality and self-related functions, including dysfunctional regulation of affect, reduced stability in personal identity, externalising behaviours, and maladaptive relationship schemas. Such characteristics are also common in individuals diagnosed with borderline personality disorder, a group with several shared risk factors and characteristics to those with DS. These findings provide further evidence for the presence of marked emotional disturbances and dysfunctional coping strategies in patients with DS. Moreover, the findings pertaining to identity impairment suggest that this sample of patients with DS experienced instability in the continuity and coherence of personal identity. This has important implications and may be linked to dissociative alterations in identity usually associated with dissociative identity disorder, for example. It seems, therefore, that examining processes relating to emotion, identity/dissociation, coping skills and relationship schemata are likely to be important for inclusion in psychological interventions for patients with DS.

### **Limitations and directions for future research**

The sample size included here was adequately powered for the design of the study. This was achieved through a pragmatic recruitment strategy which included patients who were not diagnosed on the basis of the 'gold standard' of video-EEG, in addition to those who were currently taking AEDs and/or antidepressants. Regarding diagnosis, it is unlikely that misdiagnosis was common in the present sample, because those patients diagnosed on the basis of clinical consensus had often undergone one or more other diagnostic tests, such as structural neuroimaging or routine ictal/inter-ictal EEG. Moreover, the possible effects of medication were

examined statistically, albeit with lower power than would be desirable. Larger sample sizes would provide increased power to carry out more detailed assessment of possible medication effects and/or permit rerunning analyses in patients with and without video-EEG confirmed DS.

The present study did not include a clinical comparison group. The main problem with comparing performance of patients with DS to healthy controls is that DS diagnosis is confounded with medication use, the experience of chronic seizures, mood disturbance, and/or impairment in psychosocial functioning. Whilst including a control group of patients with epilepsy might rectify some of these issues, this would not have been appropriate in this study given the potential for seizure-related neurological factors (e.g. mesial temporal sclerosis) to influence the processes under investigation (63). Other groups, such as those with mild-moderate emotional distress (anxiety, depression) or with other conversion or dissociative disorders, might be suitable control groups in future studies. Given the findings pertaining to trauma, it might also be of interest to include control groups who do and do not report previous trauma, as carried out by Roberts et al (25). Moreover, it could be valuable to examine differences in responses to facial expression stimuli in subgroups of patients with DS, such as those with and without comorbid affective, anxiety or dissociative disorders, or those with and without antecedent trauma.

Regarding experimental design, there are additional limitations to note. Due to the length of the experiment resulting from SC recording requirements, it was necessary to restrict the number of conditions to five expressions, with only one positive condition (happiness) included. Future studies might seek to balance the number of

positive and negative conditions. Moreover, response format could be varied to include additional distractor emotion labels in the response options, or perhaps to include open-ended responses.

Statistically, a relatively stringent alpha level was adopted for defining significant effects due to the large number of exploratory correlations and multiple post-hoc tests conducted. This approach was taken to present a conservative analysis, due to the large number of variables measured in a limited sample. However, some effects/correlations would have been considered significant at a lower alpha threshold (i.e.  $p < .05$ ). It is possible, therefore, that Type 2 errors may have obscured some findings in this study. Researchers may seek to recruit larger sample sizes to increase power, and/or adopt a planned comparison approach to analysis, in future studies.

## **Conclusions**

The findings presented are suggestive of an impairment in recognising emotional facial expressions in this sample of patients with DS, relative to healthy individuals. These tendencies may have developed within the context of significant traumatic life events, and associated with maladaptive relationship schemata. Elevated baseline levels of autonomic arousal in the DS group have been replicated with a novel measure (SCL) in this study, which supports previous findings. In contrast, attenuated autonomic reactions to emotional facial stimuli seem to be characteristic of some patients with the disorder.

It is likely that reduced recognition of facial expressions would lead to difficulties in interpersonal functioning in daily life; therefore, the deficit may elevate or exacerbate levels of emotional distress on an ongoing basis. As such, this difficulty could potentially increase the likelihood of seizure occurrence by contributing to elevations in affective distress, which are hypothesised to trigger individual DS. Importantly, misinterpretation of emotional signals during the process of psychological therapy might lead to undesirable difficulties in therapeutic relationships between clinicians and patients, potentially hindering therapeutic alliance and progress. As such, these findings justify further research, application in clinical contexts and possibly, the development of targeted clinical interventions aimed at improving emotional perception and interpretation.

## REFERENCES

- 1 Baslet G. Psychogenic non-epileptic seizures: A model of their pathogenic mechanism. *Seizure* 2011;20 :1-13.
- 2 Goldstein LH, Mellers JDC. Ictal symptoms of anxiety, avoidance behaviour, and dissociation in patients with dissociative seizures. *J Neurol Neurosurg Psychiatry* 2006;77 :616-621.
- 3 Moore PM, Baker GA. Non-epileptic attack disorder: a psychological perspective. *Seizure* 1997;6 :429-434.
- 4 Roberts NA, Reuber M. Alterations of consciousness in psychogenic nonepileptic seizures: Emotion, emotion regulation and dissociation. *Epilepsy Behav* 2014;30 :43-49.
- 5 WHO. World Health Organisation. The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines. Geneva: Switzerland; 1992.
- 6 American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 5th edn. (DSM-5). Washington: American Psychiatric Association; 2013.
- 7 Kihlstrom JF. Dissociative disorders. *Annu Rev Clin Psychol* 2005;1 :227-253.
- 8 Brown RJ, Cardeña E, Nijenhuis E, Sar V, Hart Ovd. Should conversion disorder be reclassified as a dissociative disorder in DSM–V?. *Psychosomatics* 2007;48 :369-378.
- 9 Harden CL. Pseudoseizures and dissociative disorders: A common mechanism involving traumatic experiences. *Seizure* 1997;6 :151-155.
- 10 Mazza M, Marca GD, Martini A, Scoppetta M, Vollono C, Valenti MA, Vaccario ML, Bria P, Mazza S. Non-epileptic seizures (NES) are predicted by depressive and dissociative symptoms. *Epilepsy Res* 2009;84 :91-96.



- 11 Bowman ES, Markand ON. Psychodynamics and psychiatric diagnoses of pseudoseizure subjects. *Am J Psychiatry* 1996;153 :57-63.
- 12 Fiszman A, Alves-Leon SV, Nunes RG, D'Andrea I, Figueira I. Traumatic events and posttraumatic stress disorder in patients with psychogenic nonepileptic seizures: a critical review. *Epilepsy Behav* 2004;5 :818-825.
- 13 Binzer M, Stone J, Sharpe M. Recent onset pseudoseizures - clues to aetiology. *Seizure* 2004;13 :146-155.
- 14 Salmon P, Al-Marzooqi SM, Baker G, Reilly J. Childhood family dysfunction and associated abuse in patients with nonepileptic seizures: Towards a causal model. *Psychosom Med* 2003;65 :695-700.
- 15 Fiszman A, Kanner AM. Comorbidities in psychogenic nonepileptic seizures: depressive, anxiety, and personality disorders. In: Schachter SC, LaFrance Jnr WC editors. *Gates and Rowan's nonepileptic seizures*. 3<sup>rd</sup> ed. Cambridge, UK: Cambridge University Press; 2010. p. 225-236.
- 16 Marchetti RL, Kurcgant D, Neto JG, von Bismark MA, Marchetti LB, Fiore LA. Psychiatric diagnoses of patients with psychogenic non-epileptic seizures. *Seizure* 2008;17 :247-253.
- 17 Reuber M, Pukrop R, Bauer J, Derfuss R, Elger CE. Multidimensional assessment of personality in patients with psychogenic non-epileptic seizures. *J Neurol Neurosurg Psychiatry* 2004;75 :743-748.
- 18 Cronje G, Pretorius C. The coping styles and health-related quality of life of South African patients with psychogenic nonepileptic seizures. *Epilepsy Behav* 2013;29 :581-584.
- 19 Goldstein LH, Drew C, Mellers J, Mitchell-O'Malley S, Oakley DA. Dissociation, hypnotizability, coping styles and health locus of control: characteristics of pseudoseizure patients. *Seizure* 2000;9 :314-322.

- 20 Testa SM, Lesser RP, Krauss GL, Brandt J. Personality Assessment Inventory among patients with psychogenic seizures and those with epilepsy. *Epilepsia* 2011;52 :E84-E88.
- 21 Bakvis P, Roelofs K, Kuyk J, Edelbroek PM, Swinkels WAM, Spinhoven P. Trauma, stress, and preconscious threat processing in patients with psychogenic nonepileptic seizures. *Epilepsia* 2009;50 :1001-1011.
- 22 Bakvis P, Spinhoven P, Zitman FG, Roelofs K. Automatic avoidance tendencies in patients with psychogenic nonepileptic seizures. *Seizure* 2011;20 :628-634.
- 23 Bakvis P, Spinhoven P, Putman P, Zitman FG, Roelofs K. The effect of stress induction on working memory in patients with psychogenic nonepileptic seizures. *Epilepsy Behav* 2010;19 :448-454.
- 24 Gul A, Ahmad H. Cognitive deficits and emotion regulation strategies in patients with psychogenic nonepileptic seizures: A task-switching study. *Epilepsy Behav* 2014;32 :108-113.
- 25 Roberts NA, Burleson MH, Weber DJ, Larson A, Sergeant K, Devine MJ, Vincelette TM, Wang NC. Emotion in psychogenic nonepileptic seizures: Responses to affective pictures. *Epilepsy Behav* 2012;24 :107-115.
- 26 Montagne B, Sierra M, Medford N, Hunter E, Baker D, Kessels RP, Haan EH, David AS. Emotional memory and perception of emotional faces in patients suffering from depersonalization disorder. *Br J Psychol* 2007;98 :517-527.
- 27 Daros A, Zakzanis K, Ruocco A. Facial emotion recognition in borderline personality disorder. *Psychol Med* 2013;43 :1953-1963.
- 28 Plana I, Lavoie M, Battaglia M, Achim AM. A meta-analysis and scoping review of social cognition performance in social phobia, posttraumatic stress disorder and other anxiety disorders. *J Anxiety Disord* 2014;28 :169-177.

- 29 Pollatos O, Herbert BM, Wankner S, Dietel A, Wachsmuth C, Henningsen P, Sack M. Autonomic imbalance is associated with reduced facial recognition in somatoform disorders. *J Psychosom Res* 2011;71 :232-239.
- 30 Schönenberg M, Jusyte A, Höhnle N, Mayer SV, Weber Y, Hautzinger M, Schell C. Theory of mind abilities in patients with psychogenic nonepileptic seizures. *Epilepsy Behav* 2015;53 :20-24.
- 31 Ponnusamy A, Marques JLB, Reuber M. Heart rate variability measures as biomarkers in patients with psychogenic nonepileptic seizures: Potential and limitations. *Epilepsy Behav* 2011;22 :685-691.
- 32 Bakvis P, Kuyk J, Spinhoven P, Roelofs K. Basal Hypercortisolism and Trauma in Patients with Psychogenic Non Epileptic Seizures. *Epilepsia* 2009;50 :170-171.
- 33 Ekman P, Friesen WV. Pictures of facial affect. Palo Alto: CA: Consulting Psychologists Press; 1976.
- 34 Dawson ME, Schell AM, Filion DL. The electrodermal system. In: Cacioppo JT, Tassinary LG, Bernston GG editors. *Handbook of psychophysiology*. 2nd ed. Cambridge: Cambridge University Press; 2000. p. 200-223.
- 35 Boucsein W, Fowles DC, Grimnes S, Ben-Shakhar G, Roth WT, Dawson ME, Filion DL. Publication recommendations for electrodermal measurements. *Psychophysiology* 2012;49 :1017-1034.
- 36 Wechsler D. Wechsler Abbreviated Scale of Intelligence (WASI); 1999.
- 37 Benton AL, Sivan AB, Hamsher KD, Varney NR, Spreen O. Contributions to neuropsychological assessment: a clinical manual; 1994.
- 38 Wechsler D. Wechsler Memory Scale - Third edition (WMS-III); 1997.
- 39 Zigmond AS, Snaith RP. The hospital anxiety and depression scale. *Acta Psychiatr Scand* 1983;67 :361-370.

- 40 Nijenhuis ERS, van der Hart O, Vanderlinden J. The Traumatic Experiences Checklist (TEC). In: Nijenhuis ERS editor. Somatoform dissociation: Phenomena, measurement and theoretical issues. Assen, Netherlands: Von Gorcum; 1999. p. 223-229.
- 41 Briere J. Inventory of Altered Self-Capacities (IASC) professional manual; 2000.
- 42 Mykletun A, Stordal E, Dahl AA. Hospital Anxiety and Depression (HAD) scale: factor structure, item analyses and internal consistency in a large population. *Br J Psychiatry* 2001;179 :540-544.
- 43 Briere J, Runtz M. The Inventory of Altered Self-Capacities (IASC): a standardized measure of identity, affect regulation, and relationship disturbance. *Assessment* 2002;9 :230-239.
- 44 Tabachnick BG, Fidell LS. Using multivariate statistics. 5th ed. Boston: Pearson Higher Education; 2007.
- 45 Faul F, Erdfelder E, Buchner A, Lang A. Statistical power analyses using G\*Power 3.1: Tests for correlation and regression analyses. *Behav Res Methods* 2009;41 :1149-1160.
- 46 Cohen J. Statistical power analysis for the behavioural sciences. 2nd ed. Hillsdale, NJ: Erlbaum; 1988.
- 47 Hall JA, Matsumoto D. Gender differences in judgments of multiple emotions from facial expressions. *Emotion* 2004;4 :201.
- 48 Biehl M, Matsumoto D, Ekman P, Hearn V, Heider K, Kudoh T, Ton V. Matsumoto and Ekman's Japanese and Caucasian Facial Expressions of Emotion (JACFEE): Reliability data and cross-national differences. *J Nonverbal Behav* 1997;21 :3-21.
- 49 Morris JS, Friston KJ, Buchel C, Frith CD, Young AW, Calder AJ, Dolan RJ. A neuromodulatory role for the human amygdala in processing emotional facial expressions. *Brain* 1998;121 (Pt 1) :47-57.

50 Williams LM, Barton MJ, Kemp AH, Liddell BJ, Peduto A, Gordon E, Bryant RA. Distinct amygdala–autonomic arousal profiles in response to fear signals in healthy males and females. *Neuroimage* 2005;28 :618-626.

51 Bakvis P, Spinhoven P, Giltay EJ, Kuyk J, Edelbroek PM, Zitman FG, Roelofs K. Basal hypercortisolism and trauma in patients with psychogenic nonepileptic seizures. *Epilepsia* 2010;51 :752-759.

52 Guinjoan SM, Bernabo JL, Cardinali DP. Cardiovascular tests of autonomic function and sympathetic skin responses in patients with major depression. *J Neurol Neurosurg Psychiatry* 1995;59 :299-302.

53 Licht CM, de Geus EJ, van Dyck R, Penninx BW. Longitudinal evidence for unfavorable effects of antidepressants on heart rate variability. *Biol Psychiatry* 2010;68 :861-868.

54 Kemp AH, Quintana DS, Gray MA, Felmingham KL, Brown K, Gatt JM. Impact of depression and antidepressant treatment on heart rate variability: a review and meta-analysis. *Biol Psychiatry* 2010;67 :1067-1074.

55 Ponnusamy A, Marques JLB, Reuber M. Comparison of heart rate variability parameters during complex partial seizures and psychogenic nonepileptic seizures. *Epilepsia* 2012;53 :1314-1321.

56 Müngen B, Berilgen MS, Arıkanoglu A. Autonomic nervous system functions in interictal and postictal periods of nonepileptic psychogenic seizures and its comparison with epileptic seizures. *Seizure* 2010;19 :269-273.

57 Opherk C, Hirsch LJ. Ictal heart rate differentiates epileptic from non-epileptic seizures. *Neurology* 2002;58 :636-638.

58 Sierra M, Senior C, Phillips ML, David AS. Autonomic response in the perception of disgust and happiness in depersonalization disorder. *Psychiatry Res* 2006;145 :225-231.

59 O'Brien FM, Fortune GM, Dicker P, O'Hanlon E, Cassidy E, Delanty N, Garavan H, Murphy KC. Psychiatric and neuropsychological profiles of people with psychogenic nonepileptic seizures. *Epilepsy Behav* 2015;43 :39-45.

60 Horley K, Williams LM, Gonsalvez C, Gordon E. Face to face: visual scanpath evidence for abnormal processing of facial expressions in social phobia. *Psychiatry Res* 2004;127 :43-53.

61 Drane DL, Williamson DJ, Stroup ES, Holmes MD, Jung M, Koerner E, Chaytor N, Wilensky AJ, Miller JW. Cognitive impairment is not equal in patients with epileptic and psychogenic nonepileptic seizures. *Epilepsia* 2006;47 :1879-1886.

62 Nicol K, Pope M, Hall J. Facial emotion recognition in borderline personality: An association, with childhood experience. *Psychiatry Res* 2014;218 :256-258.

63 Meletti S, Benuzzi F, Rubboli G, Cantalupo G, Stanzani Maserati M, Nichelli P, Tassinari CA. Impaired facial emotion recognition in early-onset right mesial temporal lobe epilepsy. *Neurology* 2003;60 :426-431.

**Table 1. Participant characteristics**

	<b>DS (n = 40)</b>	<b>Control (n = 43)</b>	<b>Test statistics</b>
Age (years)			U (83) = 806,
Median (IQR)	40 (23)	36 (20)	p = .62
Gender	Male = 8 (20%) Female = 32 (80%)	Male = 8 (18.6%) Female = 35 (81.4%)	$\chi^2$ (1, n=83) = .026, p = .87
Handedness	Right = 30 (75%)	Right = 38 (88.4%)	$\chi^2$ (1, n=83) = 2.5, p = .11
Ethnicity	White = 32 (80%) Non-white = 8 (20%)	White = 28 (65.1%) Non-white = 15 (34.9)	$\chi^2$ (1, n=83) = 2.29, p = .13
YoE			U (83) = 631,
Median (IQR)	12.5 (3)	14 (5)	p = .035
SES (NSSEC)	1 = 18 (45%) 2,3,4 or 5 = 22 (55%)	1 = 18 (41.9%) 2,3,4 or 5 = 25 (58.1%)	$\chi^2$ (1, n=83) = .083, p = .77
Taking medication	Yes (n = 29, 73%)	Yes (n = 10, 23%)	$\chi^2$ (1, n=83) = 20.2, p < 0.001
Diagnostic tests	Video-EEG (n = 27, 68%) Structural imaging		

(n = 32, 80%)	
Routine EEG (n =	
36, 90%)	
Seizure	
frequency /	
month	
Median (IQR)	4.2 (14)
Duration of	
seizure disorder	
(months)	
Median (IQR)	54 (90)
SD = standard deviation	
IQR = interquartile range	
YoE: years of full-time education (or equivalent)	
DS = dissociative seizures	
GCSE: General Certificate of Secondary Education	
AEDs = anti-epileptic drugs	
SES = socio-economic status	
NSSEC: National Statistics Socio-economic Classification system	
1 = Higher managerial, administrative and professional occupations	
2 = Intermediate occupations	
3 = Small employers and own account workers	
4 = Lower supervisory and technical occupations	
5 = Semi-routine and routine occupations	
EEG = electroencephalography	



**Table 2. Cognitive tests**

	<b>DS</b>	<b>Control</b>	<b>Test statistics</b>
<b>WASI</b>	n = 40	n = 43	
FSIQ (Mean, SD)	103.6 (14.5)	108.1 (13.1)	t (81) = 1.5, t (81), p = .14
Vocabulary (Mean, SD)	51.6 (11.1)	55.2 (9.8)	t (81) = 1.6, p = .12
Matrix Reasoning			
Median (IQR)	54 (10)	56 (15)	U (83) = 746, p = .29
<b>BFRT (all participants)</b>	n = 39	n = 43	
Median (IQR)	47 (7)	49 (5)	U (82) = 635, p = .056
<b>BFRT (minus scores &lt;40)</b>	n = 35	n = 42	
(Median (IQR))	49 (7)	49 (5)	U (77) = 598.5, p = .16
<b>WMS-III</b>			
Faces I scaled scores	n = 39	n = 43	
(Mean, SD)	10.9 (3.2)	11.1 (2.9)	t (80) = .285, p = .78

DS = dissociative seizures      SD = standard deviation

IQR = interquartile range      WASI = Wechsler Abbreviated Scale of Intelligence

FSIQ = Full-scale Intelligence Quotient      BFRT = Benton Facial Recognition Test

WMS-III = Wechsler Memory Scale – Third Edition

**Table 3. Self-report questionnaires**

	<b>DS (n = 39)</b>	<b>Control (n = 43)</b>	<b>Test statistics</b>
<b>TEC</b>			
Total (0-29; mean, SD)	8.33 (4.67)	5.69 (3.92)	t (80) = -2.12, p = .037
Impact (1-5; mdn, IQR)	4.2 (1.1)	3.6 (1)	U (83) = 493, p = .002
<b>HADS</b>			
Dep (0-21; mdn, IQR)	6 (7.5)	2 (4)	U (83) = 266.5, p < .001
Anx (0-21; mean, SD)	9.7 (3.9)	5.3 (3.2)	t (81) = -5.58, p < .001
<b>IASC</b>			
<b><i>Interpersonal Conflict</i></b>			
Mean (SD)	71 (15.8)	63.9 (12.5)	t (78) = -2.21, p = .03
<b><i>Idealisation-</i></b>			
<b><i>Disillusionment</i></b>			
Median (IQR)	68 (24.5)	58 (17)	U (80) = 662.5, p = .19
<b><i>Abandonment Concerns</i></b>			
Median (IQR)	67 (36)	51 (16)	U (80) = 457, p = .001
<b><i>Identity Impairment</i></b>			
Median (IQR)	72 (31.5)	54 (17)	U (80) = 466, p = .001
<b><i>Susceptibility to</i></b>			
<b><i>Influence</i></b>			
Median (IQR)	62 (29)	59 (17)	U (80) = 733.5, p = .55
<b><i>Affect Dysregulation</i></b>			
Median (IQR)	77 (34.5)	56 (22)	U (80) = 348, p < .001
<b><i>Tension Reduction</i></b>			

---

**Activities**

Median (IQR)

68 (38)

57 (19)

U (80) = 465, p = .001

---

TEC = Traumatic Experiences Checklist

HADS = Hospital Anxiety & Depression Scale

IASC = Inventory of Altered Self-Capacities

Anx = anxiety

Dep = depression

SD = standard deviation

IQR = interquartile range

Mdn = median

**Table 4. Facial affect recognition and SC measures**

	<b>n</b>	<b>DS</b>	<b>Control</b>
<b>Recognition</b>	DS = 38		
<b>accuracy</b>	Control = 41		
<b>(mean, SE)</b>		4.85 (0.08)	5.21 (.075)
<b>Intensity rating</b>	DS = 40		
<b>(mean, SE)</b>	Control = 43	4.6 (.197)	4.71 (.191)
<b>SCLs (<math>\mu</math>S; mean, SD)</b>	DS = 40		
	Control = 39		
<b>Baseline</b>		5.97 (5.53)	5.55 (4.64)
<b>Task</b>		7.73 (6.11)	7.14 (5.38)
<b>Percentage of</b>	DS = 40	Anger: 28.6 (25.7)	Anger: 31.8 (21.4)
<b>trials with positive</b>	Control = 39	Disgust: 23.7 (21.3)	Disgust: 23.03
<b>SCRs (0-100%)</b>		Fear: 23.3 (20.6)	(21.8)
<b>(mean, SD)</b>		Happiness: 25.3 (19.5)	Fear: 27.9 (20.1)
		Neutral: 28.7 (20.6)	Happiness: 29.9
			(24.2)
			Neutral: 32.5
			(24.3)
<b>Characteristics of</b>	DS = 40		
<b>'autonomic</b>	Control = 39		
<b>responders'</b>			
<b>N (%)</b>		16 (40)	16 (41)
<b>Male/female (%)</b>		3/13 (19/81)	3/13 (19/81)
<b>Age (years, SD)</b>		37.5 (12.3)	36.4 (12.7)

<b>SCR amplitudes –</b>	DS = 16	Anger: .232 (.238)	Anger: .607 (.822)
<b>‘autonomic</b>	Control = 16	Disgust: .363 (.376)	Disgust: .514
<b>responders’ (μS;</b>		Fear: .323 (.423)	(.767)
<b>mean, SD)</b>		Happiness: .164 (.147)	Fear: .71 (.859)
		Neutral: .395 (.359)	Happiness: 1.05
			(1.4)
			Neutral: .528
			(.517)

SE = standard error

SD = standard deviation

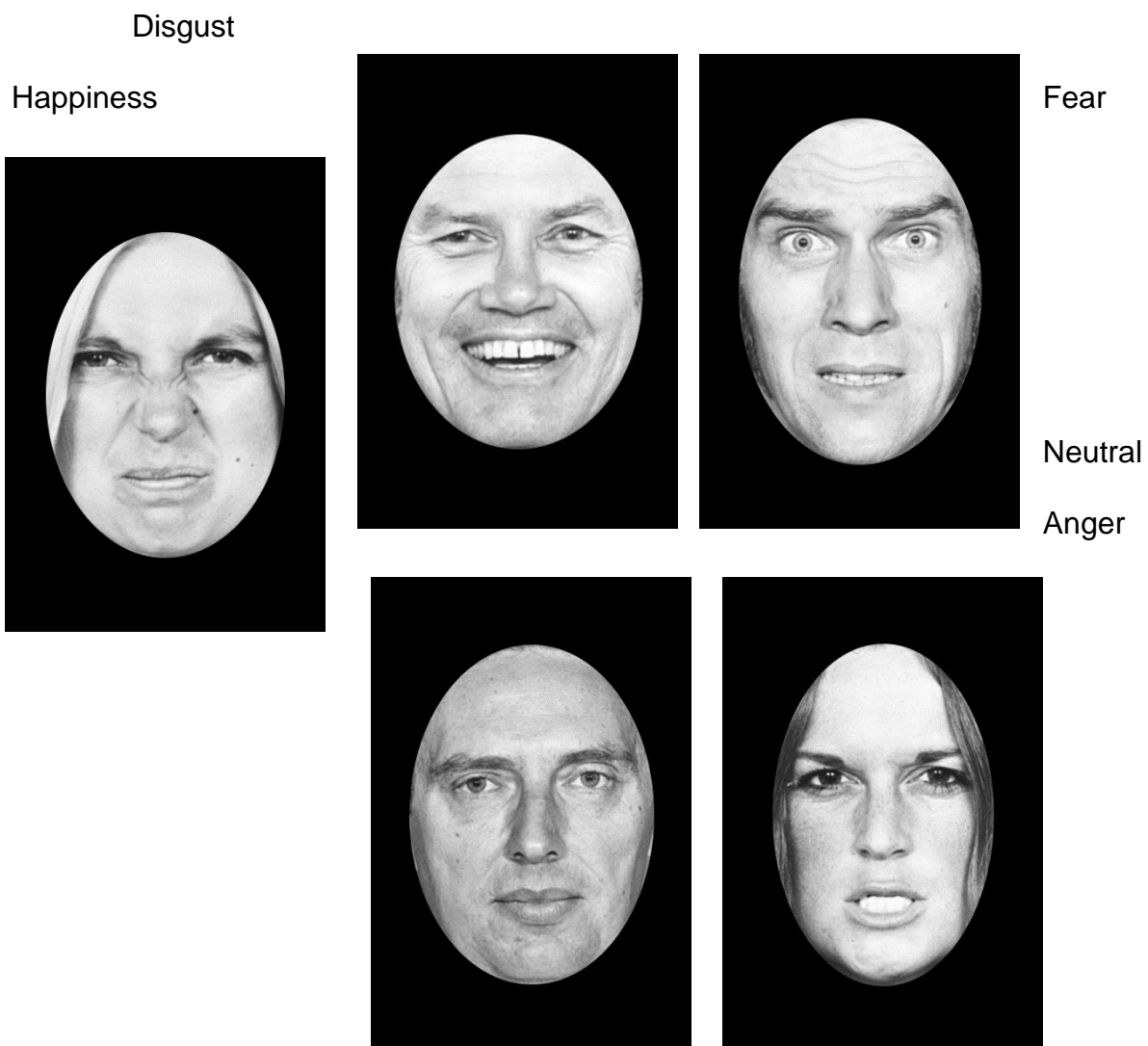
SCR = skin conductance response

μS = microSiemens

SCL = skin conductance level

## SUPPLEMENTAL DIGITAL CONTENT 1 – Details of facial stimuli

### Example stimuli



**Full list of facial stimuli (from Ekman & Friesen, 1976)**

Practice items: 022, 027, 028

Experimental items: 014, 016, 018, 020, 021, 034, 037, 038, 040, 041, 057, 059,  
061, 064, 065, 085, 088, 089, 091, 092, 093, 095, 096, 098, 099, 101, 104, 105,  
108, 110

## **SUPPLEMENTAL DIGITAL CONTENT 2 - Skin conductance measures: acquisition, extraction and reduction**

SC data were gathered using a Powerlab data acquisition system and recorded online with LabChart software (v6.0, ADInstruments). Stainless steel field electrodes were used to obtain SC recordings. Participants were requested to avoid smoking and consuming caffeine in the hour preceding the start of the session. All participants completed the experiment during the early afternoon (approximately 12-1pm), and the room temperature was held constant at approximately 20-22 degrees centigrade.

The SC electrodes were placed on the distal phalanges of the index and middle fingers of the non-dominant hand. A constant voltage (22mV<sub>rms</sub>) was applied. The SCR signal was sampled at 100 Hz. A 1Hz (second-order, low-pass) filter was applied to reduce noise in the signal. The signal was calibrated for each participant, in order to detect a range from 0-50 microSiemens (μS).

After the SC electrodes were attached, participants were asked to sit quietly during a five-minute habituation period, during which time skin conductance levels (SCLs) were measured continuously. The SC traces were visually inspected for obvious noise and/or artefact. Any portions of data that included clear noise/artefact were excluded from the analysis. Mean SCL at baseline was calculated from the average of the values obtained during the five-minute pre-experiment resting/habituation period. Mean task SCLs were calculated from the average values obtained during the final five seconds of each inter-stimulus interval (ISI). The final 5-seconds of each 15-second ISI were used for these calculations due to the fact that stimulus-driven phasic



SCRs and movement artefact produced by the manual key-press ratings confounded SCLs during the earlier section of each ISI and during stimulus-presentation. In measuring tonic SC levels, it is recommended to remove any such confounding influence (1).

For skin conductance responses, baseline values for each stimulus were calculated from the mean values during the one-second immediately prior to stimulus onset. One-second pre-stimulus onset baselines were used to ensure that any previous movement artefact/SCR did not influence the baseline measurement, and so that as little SCL drift as possible influenced comparison to the phasic SCR. The maximum SCR value occurring between one and four seconds after stimulus onset was then taken as the peak SCR amplitude for each trial. Any trials in which there was a post-stimulus decrease in amplitude were assigned a value of  $0\mu\text{S}$ . Responses of  $.01\mu\text{S}$  or greater were coded as a positive SCR.

These procedures follow well-accepted guidelines in SCR measurement (1, 2). The experimental stimuli were presented for 6-seconds each, which is a standard length of time in this type of measurement. Longer stimulus presentations would have lengthened the experiment excessively and may have resulted in disengagement from the task, particularly due to the additional necessity of relatively long baseline periods between every experimental stimulus (15-seconds) to ensure non-overlapping SCRs.

The first second of the 6-second stimulus presentation is discounted due to the rise time of SCRs – any SCRs observed within one second would be likely to have been caused by non-stimulus events or artefact (i.e. ‘non-specific SCRs’). Furthermore,

SCRs observed after approximately 4-seconds are more likely to be the result of other, undefinable, psychological processes which are not amenable to experimental control or definition. These non-specific SCRs were not considered to be informative in this particular study.

The amplitude of SCRs for a given stimulus type are the mean of the positive responses to that stimulus type, excluding values of zero (2). Positive amplitude values were averaged for each facial expression type, by participant. These values were then used to calculate average SCR amplitudes for each facial expression type, by group. Furthermore, the percentage of valid trials (i.e. trials not including noise/artefact) in which a positive SCR occurred was calculated for each participant by facial expression type, and averaged by group.

## References

- 1 Boucsein W, Fowles DC, Grimnes S, Ben- Shakhar G, Roth WT, Dawson ME, Filion DL. Publication recommendations for electrodermal measurements. *Psychophysiology* 2012;49 :1017-1034.
- 2 Dawson ME, Schell AM, Filion DL. The Electrodermal System. In: Cacioppo JT, Tassinary LG, Bernston GG editors. 2nd ed. Cambridge: Cambridge University Press; 2000. p. 200-223.



Emotion recognition in dissociative seizures

## **SUPPLEMENTAL DIGITAL CONTENT**

**SUPPLEMENTAL DIGITAL CONTENT 1- Details of facial stimuli**

**SUPPLEMENTAL DIGITAL CONTENT 2 - Skin conductance measures:  
acquisition, extraction and reduction**